

in January 2014, complete remission was sustained. However, a right side submandibular mass of about 2×2 cm was noted incidentally in June 2013. Computed tomography revealed a cystlike lesion. The patient and family refused further evaluation of the mass. The size of the mass was unchanged during 6 months of follow-up.

IAC has the advantage of delivering a high concentration of drug to the lesion. Studies by Harker and Stephen³ showed that a greater concentration of 5-FU in the tumor region is achieved by intra-arterial than by intra-venous administration. In studies by Claudio et al⁴ after IAC, 87.5% of the carcinoma of the facial skin attained resectability. However, none of the cases was an adenocarcinoma. IAC has potential for the treatment of refractory neoplasms, and may have advantages in terms of cosmetic and functional preservation.

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Resolution of urticarial vasculitis after treatment of neurocysticercosis

To the Editor: Urticarial vasculitis is most often idiopathic, but may occur in association with autoimmune disease, malignancy, drugs, or infection. Parasitic infection is a rare cause of urticarial vasculitis.¹ We report a case of urticarial vasculitis that resolved after the diagnosis and treatment of neurocysticercosis.

A 36-year-old Ecuadorian agricultural farmer who immigrated to the United States 20 years ago presented with new-onset afebrile seizure. Brain magnetic resonance imaging (MRI) revealed a ring enhancing cystic lesion (Fig 1). He also complained of a 3- to 4-month history of intermittent pruritic "rash" over his back, chest, and arms associated with a "burning" sensation and "darkening" of skin. On day 2 of hospitalization he reported worsening pruritus and new lesions on his thighs.

On exam he had partially blanching, edematous plaques and scattered erythematous papules on both posterior thighs. Side lighting revealed urticarial papules on his thighs and trunk. Hypopigmented macules were present in a linear distribution on the dorsal upper extremities, with extensive excoriations. Skin biopsy of a new urticarial papule demonstrated early leukocytoclastic vasculitis (Fig 2), leading to a diagnosis of urticarial vasculitis. Active urticarial lesions persisted for 4 days.

Labs showed a white blood cell count of $12 \times 10^9/L$, normal platelets and complement levels and 0% eosinophils. Serum antibody testing for toxoplasmosis, echinococcosis, cysticercosis, trichinellosis, histoplasmosis, human immunodeficiency virus, syphilis, and hepatitis was negative. Cerebrospinal fluid contained 6 white blood cells (83% lymphocytes) and undetectable levels of cysticercosis IgG antibodies with enzyme-linked immunosorbent assay (ELISA). Neurosurgical removal of the cystic lesion demonstrated hyalinized acellular material composed of focal parasitic remnants, consistent with the scolex of deceased parasite. The findings of parasite on brain biopsy, cystic lesions seen on MRI, and seizure fulfilled absolute, major, and minor criteria, respectively, for the diagnosis of neurocysticercosis.² The patient was started on albendazole (400 mg twice daily) and his urticarial vasculitis resolved within a few days. Two years later he remained seizure-free and rash-free.

Neurocysticercosis is a leading cause of acquired epilepsy worldwide.² Following infection, the cysticerci of *Taenia solium* remain viable in the brain and elicit minimal inflammation.³ Only after several years when a viable cyst degenerates may the

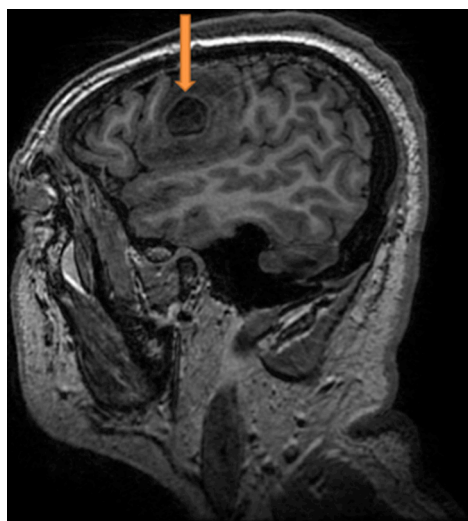


Fig 1. Neurocysticercosis in a patient with urticarial vasculitis: brain magnetic resonance imaging shows a 2.5-cm rim enhancing degenerating cyst (arrow) in the left frontal lobe.

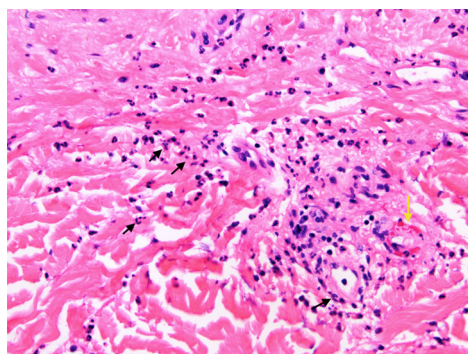


Fig 2. Urticarial vasculitis in a patient with neurocysticercosis seen on skin biopsy specimen from a new urticarial papule on the left posterior thigh. Extravasated erythrocytes (yellow arrow) and a perivascular infiltrate composed of neutrophils with neutrophilic debris (black arrows), with some mononuclear cells around the blood vessels, are consistent with an early leukocytoclastic vasculitis. (Hematoxylin-eosin stain; original magnification: $\times 40$.)

parasite induce an immune-mediated inflammatory response.³ The enzyme-linked immunoelectrotransfer blot of serum (not obtained) is the serologic test of choice, with higher sensitivity and specificity than ELISA; however, both tests are unreliable in patients with 1 degenerating cysticercus (as in our patient) or calcified granulomas.³

Urticarial vasculitis is an uncommon clinicopathologic entity distinct from acute urticaria based on dermatopathologic findings of vasculitis. Clinically, urticarial vasculitis presents with painful pruritus and erythematous wheals lasting longer than 24 hr,

followed by a residual hyperpigmentation.⁴ Our patient's differential diagnosis also included *Cysticercus cellulosae* cutis (painless subcutaneous nodules with larval cysts on biopsy)³ and Sweet syndrome (associated with pyrexia and neutrophilic infiltrate on histology without evidence of leukocytoclastic vasculitis).⁵ However, biopsy results combined with urticarial lesions on exam solidified a diagnosis of urticarial vasculitis.

To our knowledge, urticarial vasculitis associated with *T. solium* infection in which the urticarial vasculitis resolved after neurosurgical removal of the cyst and treatment with albendazole has not been reported. Physicians should continue to look for obscure causes of urticarial vasculitis.

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Ulcerations within striae distensae associated with bevacizumab therapy

To the Editor: We present the case of a 29-year-old woman with glioblastoma multiforme (GBM) who